EXPERIMENTAL STUDIES ON ARTIFICIALLY INDUCED VENTRICULAR FIBRINLLATION AND CARDIAC ARREST FOR OPEN HEART SURGERY UNDER DIRCT VISION

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EXPERIMENTAL STUDIES ON ARTIFICIALLY INDUCED VENTRICULAR FIBRILLATION AND CARDIAC ARREST FOR OPEN HEART SURGERY UNDER DIRECT VISION

by

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I. INTRODUCTION

In open heart procedure under direct vision, artificially induced ventricular fibrillation (SENNING,[16] GLENN and SWELL,[3] SHUMWAY and LEWIS[17]) or cardiac arrest (LAM, GEOGHEGAN and LEPORE[8]) has some advantages such as prevention of coronary air embolism and acquisition of a quiet operative field. Moreover, by these means ventricular fibrillation which is often provoked by cardiac approach under hypothermia, may be left out of consideration.

Accordingly these matters are now investigated from every aspect. As to the metabolism of the heart, SENNING asserted no significant difference in the rate of oxygen consumption between the beating and fibrillating heart while perfusing the body with an extracorporeal circuit. While PAUL, THEILEN and GASTEN[12] found that coronary artery-coronary sinus oxygen difference and the left ventricular oxygen consumption were decreased in the perfused fibrillating heart, as compared with the beating heart. JARDETZKY, GREENE and LORBER,[5] however, demonstrated an incresed oxygen consumption during fibrillation of the isolated dog heart. Thus considerable debate exists as to whether the heart consumes more oxygen fibrillating than beating normally. Certainly cardiac arrest provoked by chemical means would be ideal for these purposes, if it could be perfectly controlled, but the cardiac resuscitation following the completion of the surgical procedure is questionable.

The present study has been carried out to clarify whether such induced ventricular fibrillation and cardiac arrest can be used practically for intracardiac surgery

under direct vision.

II. METHODS

Healthy mongrel dogs weighing about 10kg, were deeply anesthetized with sodium pentobarbital (about 40mg/kg. intravenously). Lung inflation was maintained by intermittent positive pressure with pure oxygen through an endotracheal tube. Some animals were used under normothermia. The others were cooled by immersion in ice water except their heads until a rectal temperature of 30°C was obtained. It required usually about 30 minutes to cool the animals. The smallest quantity of sodium pentobarbital that would prevent shivering was used. After removal from the water bath the body temperature dropped to 28°C or further to 26°C, where it stabilized. With sterile technique, the right chest was entered through the fifth intercostal space except for the group in which a left atriotomy was made. For varying periods of time, the methods of cardiac occlusion, which will be mentioned below, were undertaken and in some of the hypothermic dogs, furthermore three kinds of cardiac approach, namely, a right atriotomy, a right ventriculotomy and a left atriotomy, were performed. And the results of operation under every method of cardiac occlusion, were compared with each other. In suturing a cardiotomy, a physiological saline solution was sufficiently poured into the cardiac cavity through the incision to drive bubbles and prevent air embolism.

The methods of cardiac occlusion were as follows.

(i) Ventricular fibrillation was induced by electric stimulus of 10 volts for one second, through the heart which was placed between both electrodes, metal plates of 5 cm in diameter. But the left and right auricles were beating in normal rhythm. It was decided that the cardiac occlusion began to be raised at that monent.

(ii) Cardiac arrest was induced by KCl solutions of different concentrations, as it will be mentioned below, being poured into the left ventricle. After a while, both ventricles stopped their activities in the state of dilatation. At the same time, the left auricle also stopped its pulsation perfectly, but the right held its normal rhythm.

(a) As soon as a 5% KCl solution was poured into the left ventricle, the ascending aorta was clamped and the heart was manually squeezed, in order to promote rapidly coronary perfusion of the solution. For this purpose 0.67cc of the solution pro kg of body weight, on an average, was required.

(b) Instilment of a 20% KCl solution into the left ventricle. In this case, the blood stream through the ascending aorta was not stopped. On an average, 0.18cc of the solution pro kg of body weight induced cardiac arrest.

(iii) Inflow and outflow occlusion was performed as the control of the above mentioned methods of cardiac occlusion. The azygos vein was ligated permanently and the superior, then the inferior vena cave was occluded. After a few beats were allowed for cardiac emptying, the aorta and pulmonary artery were simultaneously occluded. Release from the occlusion was practised in the same order. At the time of a left atriotomy (a left thoracotomy), only the outflow was closed.

For 2 minutes bofore the above mentioned cardiac occlusions were carried out, positive pressure breathing was securely replaced with positive-negative pressure one, and also after release from the occlusions, it was continued until the cardiac activity was perfectly recovered to the normal state. In the hypothermic dogs, rewarming with steam heat was started as soon as cardiac resuscitation was begun and continued until a rectal temperature of 35°C was obtained, where spontaneous breathing appeared vigorously. At that time an intratracheal tube was removed.

As cardiac resuscitation, after the completion of the surgical procedures, following methods were used.

(i) At first, cardiac massage was started until the myocardium became pink. By this means, the ventricular fibrillation was altered from a state of atony to a convulsive phase and the cardiac arrest to a phase of coarse fibrillation, ready for the defibrillating electric shock. In the cases of inflow and outflow occlusion, when ventricular fibrillation spontaneously occured, cardiac resuscitation was carried out by the same means.

(ii) Countershock was performed to convert the heart to the regular rhythm. Three successive electric stimuli, whereof each was 130 volts for 1/20 seconds, were regarded as one countershock. If unsuccessful at one time, cardiac massage and coutershock were alternately repeated.

When the heart did not regain sufficiently its tonus by simple massage, the following managements were undertaken.

(iii) After instilment of 1 : 10,000 epinephrine 1cc or a 10% $CaCl_2$ solution from 1 to 2cc into the left ventricle, massage and then shock were performed. Noticing that epinephrine was more effective than the $CaCl_2$ solution, we used principally only epinephrine in the later half of experiments.

To all the experimental dogs, penicillin solution of 200,000 units was poured into the thoracic cavity on the side of operation. The chest wall was firmly closed with two layers suture and no drainage was practised.

Not to mention when the dogs died, but even when survived, they were sacrificed in various periods. And the parts of both ventricular walls were resected, fixed in a 10% formalin solution and stained by hematoxylin-eosin. These microscopic preparations were investigated histologically.

Table 1. Results of Various Kinds of Cardiac Occulusion under Normothermia

Cardiac Occlusion		Occlusion Time (Minute)	Number of Dogs	Immediate Death	Late Death	Long Term Survival	Cerebral Damage in Survivals
Ventricular Fibrillation		2	1	0	0	1	0
		3	2	0	0	2	0
		4	**3**	**1**	**0**	**2**	**1**
		4.5	1	0	0	1	0
		5	1	1	0	0	
Cardiac Arrest	5% KCl	**4**	**2**	**0**	**0**	**2**	**1**
		4.5	3	1	1	1	0
		5	1	1	0	0	
	20% KCl	2.8	1	0	0	1	0
		4	**3**	**1**	**1**	**1**	**0**
		4.5	1	0	1	0	
Inflow and Outflow Occlusion		**4**	**1**	**0**	**0**	**1**	**0**
		4.5	1	0	0	1	0
		5	2	1*	0	1	1
		6	2	0	1	1	1

Immediate Death : the dogs died soon after the completion of the surgical procedures, regardless of whether cardiac resuscitation was performed successfuly or not

Late Death : the dogs survived the procedues, but died in a short period without complete recovery.

Long Term Survival : the dogs recovered completely and were sacrificed some days later. But in this group, a few dogs which were emaciated gradually and died from cerebral damage or pyothorax, were included.

*……The dog died due to some troubles of anesthesia apparatus.

III. RESULTS

1. Comparison of various kinds of cardiac occlusion under normothermia (25 dogs)

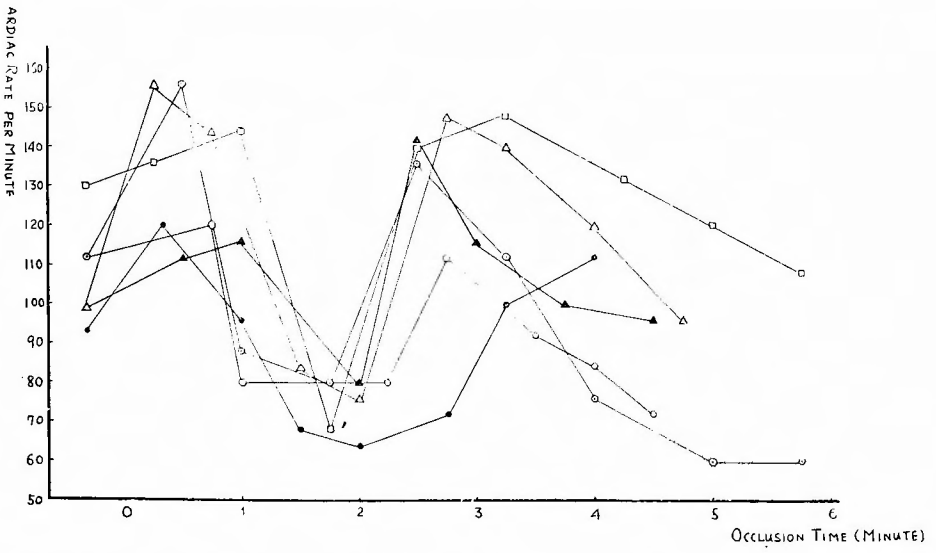
As shown in Table 1, (a) ventricular fibrillation, (b) cardiac arrest and (c) inflow and outflow occlusion were carried out for various periods under normothermia. We obtained the following results.

(i) In any method of cardiac occlusion, the limit of occlusion time, in which the dogs were able to be alive for a long term without any affection, was about 4 minutes. Even in such an occlusion time, however, cerebral damages were raised in some cases. One case of ventricular fibrillation was affected with convulsions and lost its eyesight and died 6 days after the operation. The other case of cardiac arrest had spastic paralysis and died 13 days after the operation.

(ii) In the method (c) the heart regained most easily its normal activity after release from occlusion, compared with the results in (a) and (b). Even through inflow and outflow occlusion for 5 or 6 minutes, the heart was possible to recover, but cerebral damages (spastic paralysis and loss of eyesight), of which two dogs died several days after the operation, developed. One case, which was subjected to inflow and outflow occlusion for 5 minutes, died just after the operation due to some troubles of anesthesia apparatus.

(iii). When cardiac occlusion was carried out by the method (c). as shown

Fig, 1. Changes of Cardiac Rate Following Inflow and Outflow Occlusion in Normothermic Dogs



in Fig. 1, just after occlusion, cardiac rate increased rapidly, after about one minute decreased contrarily, after 2 minutes and 30 seconds increased again and after 3 minutes and 30 seconds decreased slowly. In the course of time heart beat became weak, but none among 6 cases had ventricular fibrillation during

occlusion for 4 to 6 minutes. After release, cardiac resuscitation was unnecessary at all or cardiac massage for only short time was needed.

(iv) In the method (b), by using a 5% KCl solution better results were obtained, because cardiac arrest was apt to rise again even after cardiac resuscitation was achieved successfuly, when a 20% KCl solution was used.

(v) By any method of cardiac occlusion, the pupils which had been 1 to 2 mm in diameter, were started to dilate after 5 to 7 seconds and reached to the maximum after 1 to 3 minuts (1 minute and 40 seconds on an average). These dilated pupils were gradually contracted again by cardiac resuscitation. However, the cases, of which the pupils were unable to be contracted or dilated again after transitory contraction, were absolutely bad in prognosis, even when heart beat was returned to the normal rhythm.

2. Comparison of various kinds of cardiac occlusion under hypothermia (56 dogs)

(A) Results of cardiac occlusion for 15 minutes under hypothermia Under hypothermia (28°C to 26°C), the three kinds of cardiac occlusion for 15 minutes were performed and their results were compared with each other.

(i) As shown in Table 2, inflow and outflow occlusion had the lowest mortality.

Table 2. Results of Various Kinds of Cardiac Occlusion for 15 Minutes under Hypothermia

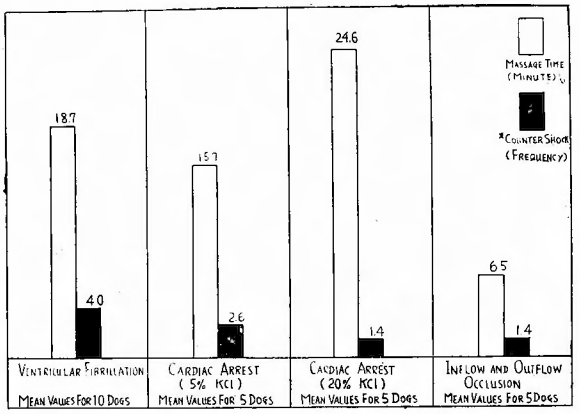
Cardiac Occlusion	Number of Dogs	Immediate Death	Late Death	Long Term Survival	Mortality
Ventricular Fibrillation	10	4	1	5	50%
Cardiac Arrest { 5% KCI	5	2	0	3	40%
Cardiac Arrest { 20% KIC	5	0	3	2	60%
Inflow and Outflow Occlusion	5	1	0	4	20%

(ii) Between the two methods of ventricular fibrillation and cardiac arrest, no significant difference was found in view of the results. In the latter cases, however, as in the normothermic dogs, the application of a 5% KCl solution had better results than a 20% KCl solution.

(iii) The relation between each kind of cardiac occlusion and cardiac resuscitation was shown in Fig. 2. The cases of inflow and outflow occlusion reacted most rapidly to cardiac resuscitation, namely, the heart regained its activity after cardiac massage of the shortest duration and the minimum frequencies of countershock (three successive electric stimuli regarded as one countershock).

(iv) The cardiac arrest induced by a 20% KCl solution was converted to the regular rhythm by means of less countershocks and cardiac massage of shorter duration than the ventricular fibrillation induced by electric stimulus. But in the cases of a 5% KCl solution, cardiac massage for the longest time was necessary, despite of the same frequencies of countershock as in the cases of inflow and outflow occlusion.

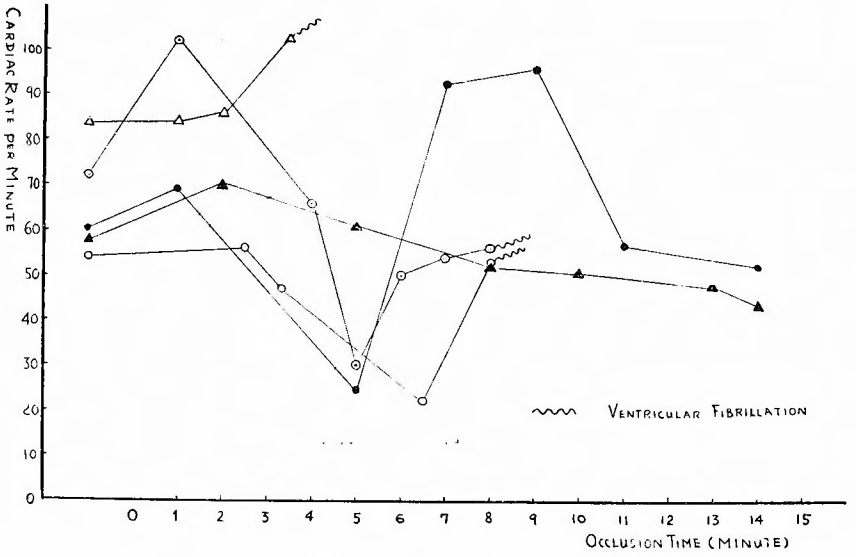
Fig. 2. Frequencies of Countershock and Periods of Cardiac Massage Needed for the Accomplishment of Resuscitstion Following Various Kinds of Cardiac Occlusion for 15 Minutes under Hypothermia



*……The electric stimuli for successive 3 times were regarded as one countershock.

(v) As shown in Fig. 3, in 3 of 5 cases applied with inflow and outflow occlusion, ventricular fibrillation developed spontaneously, but it was resuscitated by means of cardiac massage for 5 to 6 minutes in amount and countershock of 1 to 2 times. In one of the remaining 2 cases without ventricular fibrillation, changes of cardiac rate in the course of time showed the similar curve like that under normothermia. In the other one, however, cardiac rate which had been increased just after occlusion, was slowly decreased in the course of time, showing a flat curve. In the former, no cardiac resuscitation

Fig, 3. Changes of Cardiac Rate Following Inflow and Outflow Occlusion for 15 Minutes under Hypothermia



was needed, but in the latter, cardiac arrest occured just after release from occlusion and then was converted to the regular rhythm. But the dog died without appearance of spontaneous breathing even after rewarming to a rectal temperature of 34°C.

(vi) Under hypothermia, in any method of cardiac occlusion the pupils of all animals began to dilate from 1 to 2 minutes later. They dilated extremely after 2 minutes and 30 seconds to 6 minutes (on an average 4 minutes and 22

seconds) from occlusion and were contracted slowly to the original size by means of cardiac resuscitation. The cases, of which the pupils were not contracted or dilated again after contraction were absolutely bad in prognosis.

(B) Results of cardiac approaches under hypothermia

In hypothermic dogs, the three kinds of cardiotomy were made under the above mentioned methods of cardiac occlusion for 4 to 9 minutes. At that time, a 5% KCl solution was only used to induce cardiac arrest, because it was superior to a 20% KCl solution in results.

Table 3 Results of the Cardiotomies under the 3 Kinds of Occluding Method for 4 to 9 Minutes in Hypothermic Dogs

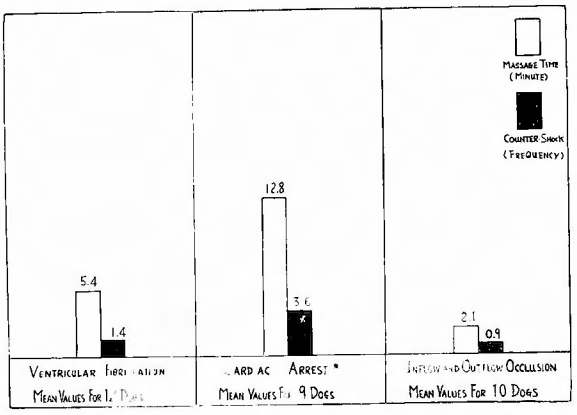
Cardiac Occlusion	Cardial Approach	Number of Dogs	Immediate Death	Late Death	Long Tern Survival	Mortality
Ventricular Fibrillation	L. Atriotomy Inspection of the Mitral Valve	4	0	0	4	25%
	R. Atriotomy	4	0	1	3	
	R. Ventriculotomy	4	0	2	2	
Cardiac Arrest (5% KCl)	L. Atriotomy Inspection of the Mitral Valve	3	1	1	1	44%
	R. Atriotomy	3	0	1	2	
	R. Ventriculotomy	3	0	1	2	
Inflow and Outflow Occlusion	L. Atriotomy Inspection of the Mitral Valve	3	1*	0	2	20%
	R. Atriotomy	4	0	0	4	
	R. Ventriculotomy	3	0	1	2	

*……The dog died of air embolism.

(i) As shown in Table 3, there was the lowest mortality rate in the group of inflow and outflow occlusion. One case of this group undergoing a left atriotomy died of air embolism immediately after operation, owing to insufficient instillment of a saline solution into the cardiac cavity through the incision at the time of suturing. A lower mortality rate was obtained in the group of ventricular fibrillation than in the group of cardiac arrest.

(ii) In Fig. 4 like in Fig. 2, the relation between various kinds of cardiac occlusion and cardiac resuscitation was shown. On such points of view, the cases of inflow and outflow occlusion were the most responsive to cardiac resuscitation. However, unlike the cases undergoing cardiac occlusion for 15 minutes under hypothermia, cardiac resuscitation was more easily accomplished in the cases of ventricular fibrillation than in the cases of cardiac arrest. Also judging from the fact that the mortality rate at cardiac approach was lower in the cases subjected to

Fig. 4. Frequencies of Countershock and Periods of Cardiac Massage Needed for the Accomplishment of Resuscitation Following the Cardiotomies under the 3 Kinds of Occluding Method for 4 to 9 Minutes in Hypothermic Dogs



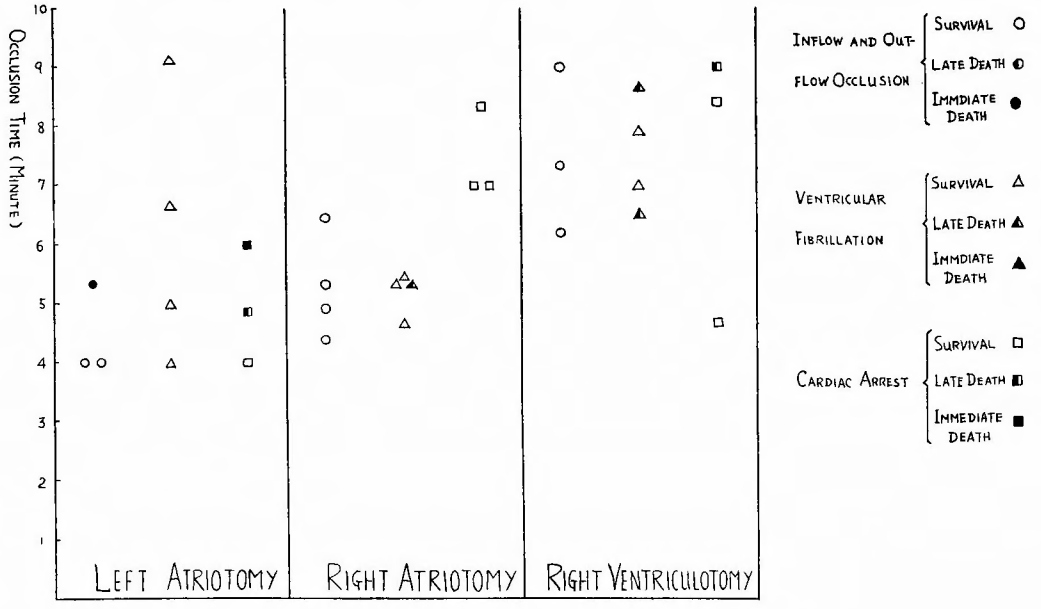
*……In inducing cardiac arrest, a 5% Kcl solution was only used.

ventricular fibrillation for 4 to 9 minutes than in those subjected to cardiac arrest of the same duration, the former was considered to be more advantageous here than the latter.

(iii) Air embolism by which cardiac approach under inflow and outflow occlusion is often followed, was completely avoided by means of sufficient instilment of a saline solution into the cardiac cavity through the cardiotomy at the time of suturing.

(iv) Even in cases of ventricular fibrillation or cardiac arrest, it was necessary to suck up a considerable quantity of blood to inspect distinctly the inner structure of the heart, unless at a right atriotomy or ventriculotomy both inflow and outflow were closed, and at a left atriotomy only outflow. Without these managements, experimental dogs would die from bleeding. Particularly, without outflow occlusion at a left atriotomy, a large quantity of arterial blood flowing back through the

Fig. 5. Relation between the Death from Operation and the Duration of Cardiac Occlusion under Hypothermia



aortic and mitral valves was lost.

(v) Fig. 5 shows the relation between the death following the cardiac approaches under the three kinds of cardiac occlusions and the duration of them. In occlusion time for 4 to 9 minutes, however, no marked relation was demonstrable.

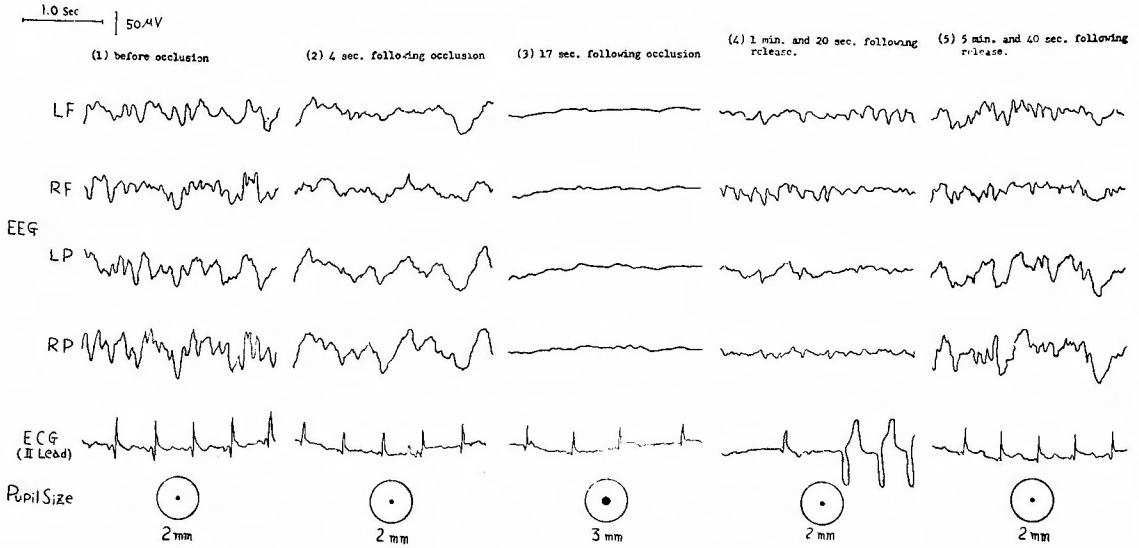
(vi) Of 10 cases undergoing various kinds of cardiotomy under inflow and outflow occlusion, one immediately after the release from and 6 during the occlusion, had ventricular fibrillation, but all were resuscitated. Only one of them died soon after the operation, but the others belonged to the group of long term survival. Of the remaining 3 cases without ventricular fibrillation, one survived for short term and the other two for long term.

3. Relation between changes of pupil size and those of electroencephalographic pattern following cardiac occlusion and release from it

Either under normothermia or under hypothermia, the pupils of all experimental dogs dilated extremely a certain time after cardiac occlusion, and then were gradually contracted by means of cardiac resuscitaion. But the cases, of which the pupils were not contracted or dilated again after transitory contraction, were absolutely bad in prognosis even though the heart regained its regular rhythm, as previously mentioned. Namely, the recovery of cardiac function became more completely in proportion to the degree of pupilar contraction. It seemed that this fact was connected with an effective cardiac output, that is to say, the degree of oxygen supply to the brain. Then the relation between changes of pupil size and those of electroencephalographic pattern following cardiac occlusion and release from it was investigated.

Four dogs were anesthetized with pentobarbital intravenously. Lung inflation was maintained by intermittent positive pressure with pure oxygen through an

Fig. 6. Changes of the Pupil Size and the Electroencephalographic Pattern Following Inflow and Outflow Occlusion and Release from It under Normothermia (dog No. 101)



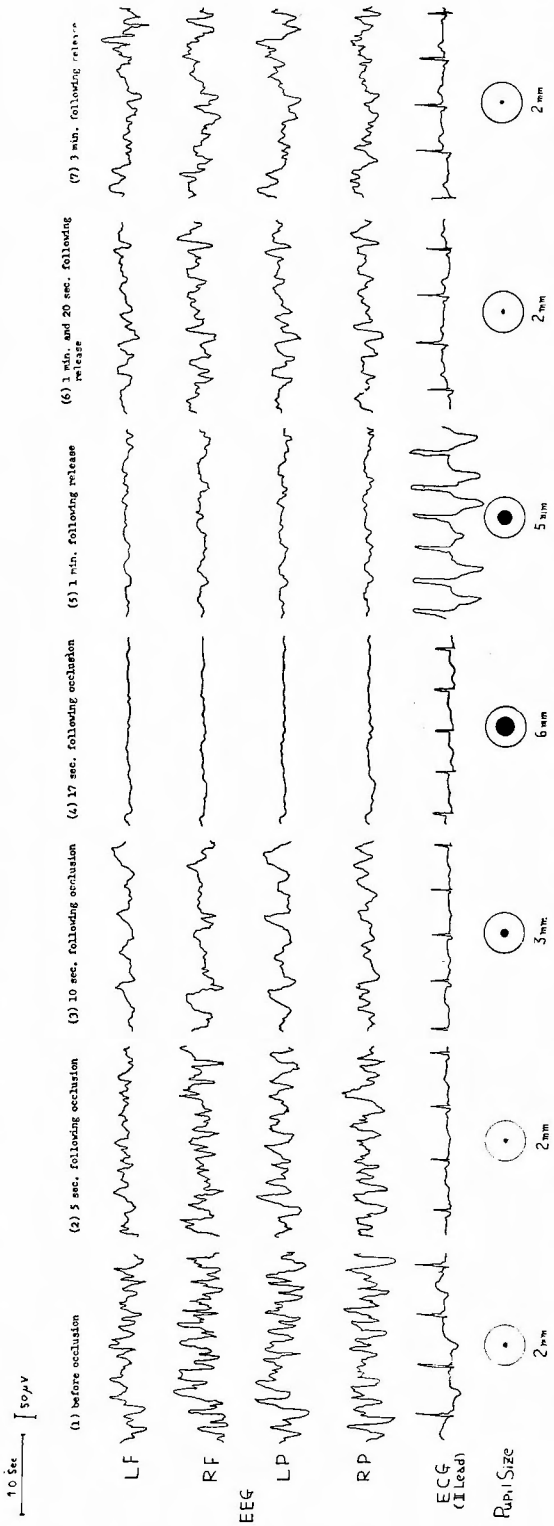


Fig. 7. Changes of the Pupil Size and the Electrooncephalographic Pattern Following Inflow and Outflow Occlusion and Release from It under Normothermia (dog No. 102)

endotracheal tube. Either under normothermia or under hypothermia, the right chest was entered through the fifth intercostal space to perform inflow and outflow occlusion. And then the above mentioned matters were researched.

(A) Cases under Normothermia

(i) No. 101 (8kg pentobarbital 240mg), Fig. 6.

Before occlusion, slow waves of 4 to 6 cycles per second activity superimposed by smaller ones of 10 to 13 cycles appeared in the electroencephalogram, and the pupils were 2 mm in diameter. Four seconds after it, the smaller became undistinguishable. Seventeen seconds after it, the waves which had been flatten, disappeared at all. At that time, the pupils were 3 mm in diameter. Afterwards, they dilated gradually to be 6 mm (22 sec.), 8 mm (1 min. and 40 sec.) and 12 mm (1 min. and 50 sec.) in diameter, where their dilatation was maximum. Forty seconds after release from occlusion for 2 minutes and 35 seconds, they began to contract and became in the course of time to be 7 mm (50 sec.), 5 mm (1 min.), 4 mm (1 min. and 10 sec.), 3 mm (1 min. and 15 sec.) and 2 mm (1 min. and 20 sec.) in diameter, where they returned to the original state. While in the electroencephalogram, 1 minutes and 15 seconds after it, low voltage slow waves of 3 to 5 cycles per second activity began to appear and a few seconds later became somewhat distinct. Afterwards, they recovered gradually to the state before occlusion in 5 minutes and 40 seconds from the moment of release.

(ii) No. 102 (5kg, pentobarbital 300mg), Fig. 7.

Before occlusion, irregular high voltage waves of 12 to 18 cycles per second activity were observed and the

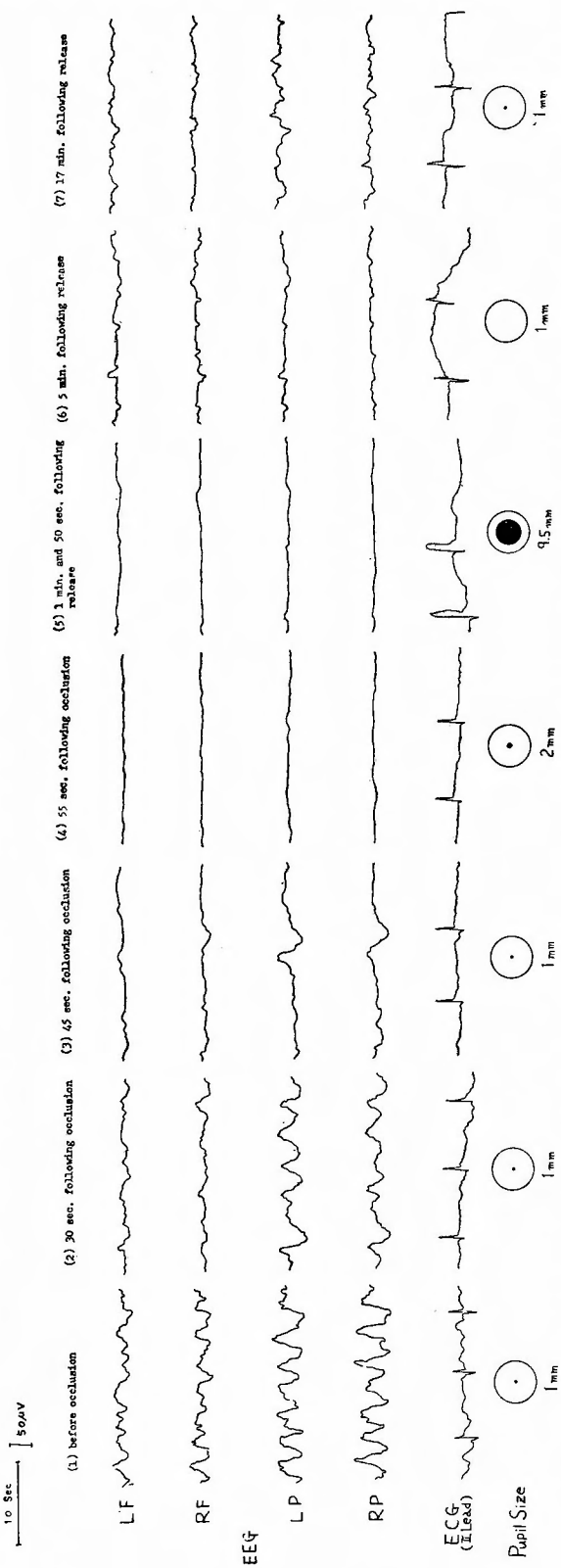


Fig. 8. Changes of the Pupil Size ane Electroencephalographic Pattern Following Inflow and Outflow Occlusion and Release from It under Hypothermia (dog No. 201)

pupils were 2 mm in diameter. Five seconds after it, they decreased in ampulitude without changing their frequency and after 10 seconds, did in frequency. At that time, the pupils were 3 mm in diameter. After 17 seconds, the electroencephalographic activity disappeared completely and the pupils dilated to be 6 mm. Afterwards, they continued to increase in diameter, reaching to the maximum; 8 mm (43 sec), 9 mm (55 sec,) and 10 mm (1 min. and 25 sec.). Thirty seconds after release from occlusion for 2 minutes, contrarily they began to contract gradually, coming up to the original size; 8 mm (36 sec.), 7 mm (44 sec.), 6 mm (49 sec.), 5 mm (54 sec.), 4 mm (1 min. and 24 sec.), 3 mm (2 min.), and 2 mm (2 min. and 15 sec.). The disappeared waves began to reappear in 1 minute after release, but even when the pupils returned to their original size, they were still in the low voltage slow wave pattern, and restored to the former condition after 3 minutes.

(B) Cases under hypothermia

(i) No. 201 (7 kg, pentobarbital 450 mg, a rectal temperature of 30°C), Fig. 8.

Before cardiac occlusion, irregular high voltage slow waves of 4 to 5 cycles per second superimposed by smaller ones of 10 to 12 cycles per second were seen, and the pupils were 1 mm in diameter. Twenty seconds after it, the electroencephalogram in both frontal regions and 30 seconds that in both parietal regions showed decrease in ampulitude and disappearance of smaller waves. After 45 seconds, the electrical activities of the various regions declined remarkably, but still no change was seen in pupil size. After 55 seconds, the activities disappeared and the pupils dilated somewhat to be 2 mm in diameter. Then, the pupils dilated gradually to come up to the

maximum; 3 mm (1 min. and 37 sec.), 4 mm (2 min. and 30 sec.), 6 mm (3 min. and 22 sec.), 7 mm (3 min. and 40 sec.), 8 mm (3 min. and 50 sec.), 9 mm (4 min. and 10 sec.) and 10 mm (4 min. and 25 sec.). After 4 minutes and 40 seconds, the occlusion was released. In 1 minute and 50 seconds after release, they began contrarily to contract. However, the electroencephalogram showed still no electrical activity, while the pupils continued gradually to contract, reaching to the original size, i. e. 7 mm (2 min. and 0 sec.) 5 mm (2min. and 10 sec.), 4 mm (2 min. and 20 sec), 3 mm (2 min. and 40 sec.), 2 mm (2 min. and 56 sec.), and 1 mm (4 min. and 5 sec.). Five minutes after release, waves of extremely low voltage began to appear, but even after seventeen minutes they were not restored to the state before occlusion.

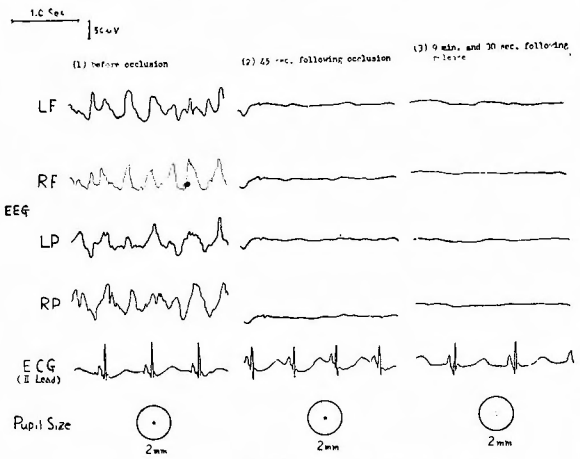


Fig. 9. Changes of the Pupil Size and the Electroencephalographic Pattern following Inflow and Outflow Occlusion and Release from it under Hypothermia (dog No. 202)

(ii) No. 202 (8 kg. pentobarbital 450 mg, a rectal temperature of 30°C), Fig, 9.

Befor occlusion, irregular slow waves of 4 to 5 cycles per second were found and the pupils were 2 mm in diameter. Afterwards, they became gradually to decrease in ampulitude and frequency until they disappeared at all 45 seconds after occlusion. At that time, the pupils had no change in their size. Then, they increased gradually in diameter, reaching to the maximum degree, i. e. 3 mm (1 min. and 25 sec.), 4 mm (1 min. and 40 sec.), 5 mm (2 min. and 10 sec.), 6 mm (2 min. and 40 sec.), 7 mm (2 min. and 50 sec.), 8 mm (3 min.), 9 mm (3 min. and 15 sec.), 10 mm (3 min. and 35 sec.), and 11 mm (4 min. and 30 sec.). After 5 minutes and 35 seconds, the occlusion was released. Forty eight seconds after release, they began to contract to be 10 mm in diameter and this change progressed in the course of time; 9 mm (1 min. and 25 sec.), 8 mm (3 min. and 28 sec.), 7 mm (3 min. and 55 sec.), 6 mm (4 min. and 30 sec.), 5 mm (5 min. and 40 sec.), 4 mm (5 min. and 52 sec.), 3 mm (6 min. and 35 sec.) and 2 mm (9 min. and 30 sec.). However, even 15 minutes after release, electrical activity was not demonstrable in the electroencephalogram.

After all, in the two normothermic dogs undergoing inflow and outflow occlusion respectively for 2 minutes and 35 seconds and 2 minutes, the electrical activities of various regions disappeared 17 seconds after occlusions, and the pupils dilated to the extreme respectively 1 minute and 50 seconds and 1 minutes and 25 seconds. After release from occlusion, respectively 1 minute and 20 seconds and 2 minutes and 15 seconds, the pupils contracted to the original sizes, while the disappeared electrical activities recovered to the former conditions in one case 5 minutes and 40 seconds and in the other 3 minutes. In the two hypothermic dogs undergoing the same occlusion respectively for 4 minutes and 40 seconds and 5 minutes and 3 seconds, after occlusion the electroencephalograms became to show no activity respectively in 55 and 44 seconds, and the pupils dilated to the extreme in 4 minutes and 25 seconds in one case and in 4 minutes and 30 seconds in the other. Respectively 4 minutes and 5 seconds and 9 minutes and 30 seconds after release the pupils of

these dogs were restored to the former size, while in the electroencephalograms, even after 17 minutes lower voltage pattern was seen in one case and after 15 minutes no electrical activity appeared in the other one.

In sum, by inflow and outflow occlusion at first electroencephalographic deterioration appeared, and then the pupils began to dilate. After electrical activities vanished at all, the pupils dilated to the maximum. After release from occlusion, electroencephalographic pattern was restored slowly, but prior to it, the pupils contracted to recover to the former size. However, it seemed, under hypothermia, to need a considerable long time for the recovery of electroencephalographic pattern.

4. Histological findings proved in cardiac tissue of the experimental dogs

The histological examination of cardiac tissue has been carried out in almost all experimental dogs to learn the method of cardiac occlusion under which the slightest cardiac damage occured among the three kinds of occluding method above mentioned.

(A) Cases under normothermia (Table 4)

The histological findings of myocardium in dogs undergoing cardiac occlusion for various periods under normothermia were summarized in Table 4. Classifying

Table 4. Histological Findings of Myocardium Resulting from Cardiac Occlusion for Various Periods under Normothermia

Cardiac Occlusion	Dog No.	Survival Time (Day)	Occlusion Time (Min.)	Massage Time (Min.)	Counter Shock (Frequency)	Myocardium						
						Deg	Nec	Cal	Gra Inf	Hem	Abn Arr	Fra
Ventricular Fibrillation	4	0	4.0	6.8	7	+	−	−	−	−	−	−
	7	0	5.0	1.2	2	−	−	−	−	−	−	−
	5	7	4.0	3.5	3	++	++	−	+	−	−	−
	8	7	4.5	0.9	1	+	++	+	−	−	−	−
	1	15	2.0	0.8	2	+	+	+	+	−	−	−
Cardiac Arrest 5% KCl	65	0	5.0	13.0	6	+++	+	−	+	+++	++	+
	66	0	4.5	10.0	6	++	−	−	−	−	++	+
	68	20(H)	4.5	22.0	7	+++	−	−	+	−	++	+
	67	10	4.0	8.0	5	++	−	−	+	−	++	+
	63	13	4.0	0.8	1	++	+	+	+	−	++	+
	64	13	4.5	1.3	1	++	−	−	−	−	++	+
Cardiac Arrest 20% KCl	12	0	4.0	8.2	3	−	−	−	−	−	−	−
	10	10(H)	4.0	1.0	1	−	±	−	−	−	−	−
	11	10(H)	4.5	6.2	7	+	+	−	++	+++	+	+
Inflow and Outflow Occulsion	74	12(H)	6.0	0.6	0	++	−	−	−	−	−	−
	73	4	6.0	0.0	0	+	++	+++	++	−	−	−
	70	5	5.0	1.8	0	±	−	−	+	−	−	−
	69	8	4.0	0.0	0	+++	+	++	−	−	++	++
	71	8	4.5	1.3	0	+	−	−	+	−	−	−

Key : Deg·····Degeneration Nec·····Necrosis
Cal·····Calcification Gra Inf····· Granulocyte Infiltration
Hem ···Hemorrhage Abn Arr ···Abnormal Arrangement
Fra ···Fragmentation (H)·····(Hours)
These symbols are available in Table 5. and 6.

the animals into three groups according to the kind of occluding method, the author arranged them in the order from case of shorter to longer term survival. This matter is available in Table 5 and 6.

(i) Group of ventricular fibrillation

In the cases of immediate death (No. 4 and 7), despite of relatively long term occlusion (respectively 4 min. and 5 min), pathological changes were not found in both epicardium and myocardium except only slight subepicardial degeneration of myocardium (homogeneous and reddish staining). But in the cases which were sacrificed 7 or 15 days after operation (No. 5, 8 and 1), epicardial thickening with granulocyte infiltration and myocardial degeneration in various degrees (disappearance of transverse striae, fusion of myoplasma and swelling of cells and nuclei), besides focal myocardial necrosis with granulocyte infiltration, proliferation of connective tissue and calcification, or disseminated small foci of coagulating muscle necrosis were found.

(ii) Group of cardiac arrest

In the cases (N. 65 and 66) which were resuscitated with difficulty and died just after the operation, marked histological changes of myocardium were found; in both cases, abnormal arrangement and fragmentation of muscle fibers with epicardial hemorrhage and thickening, besides in the former, intense hemorrhage extending between muscle fibers, myocardial degeneration (vacuolization and cloudiness of protoplasm) and disseminated small foci of muscle necrosis (Fig. 10 and 11), and in the latter, moderate myocardial degeneration (remarkable in subepicardial region). On the contrary, in the case of immediate death (No. 12) which was easily resuscitated, no histological change of cardiac tissue was proved. Also in the cases of late death (No. 68, 10 and 11), the extent of myocardial damage was in proportion to the difficulty of cardiac resuscitation. In the cases of long term survival (No. 67, 63 and 64), myocardial damages were almost equal in degree.

(iii) Group of inflow and outflow occlusion

In this group, no one had ventricular fibrillation. Three of 5 dogs were resuscitated only by cardiac massage for 0.6 to 1.8 minutes due to atony of the heart after release from occlusion, and the remaining 2 recovered without this management. In No. 74, a case of late death, epicardium was almost normal and moderate myocardial degeneration (pycnosis or swelling of nuclei, and perinuclear vacuolization) was found. The remaining four were cases of long term survival. In No. 73 and 69 recovering without cardiac massage, severe epicardial thickening with granuocytes infiltration was found. In the former, focal myocardial necrosis with calcification and inflammatory reaction and slight myocardial degeneration (mainly in subepicardial region). In the latter, the similar necrosis and more advanced degeneration were demonstrable (Fig. 12). In both No. 70 and 71, resuscitated by cardiac massage for short time, moderate epicardial thickening and slight myocardial degeneration (pycnosis and atrophy of muscle fibers) were found.

(iv) Résumé

It was a general tendency, that after same duration of the occlusion, the more

difficult cardiac resuscitation was, i. e., the longer the massage time and the more frequent the coutershock was, the more striking the pathological findings of myocardium were. Under normothermia, however, considerable myocardial degeneration and necrosis were taken place only by inflow and outflow occlusion for 4 to 6 minutes.

Table 5. Histological Findings of Myocardium Resulting from Cardiac Occlusion for 15 Minutes under Hypothermia

Cardiac Occlusion	Dog No.	Survival Times (Day)	Massage Time (Min.)	Counter Shock (Frequency)	Myocardium						
					Deg	Nec	Cal	Gra Inf	Hem	Abn Arr	Fra
Ventricular Fibrillation	18	5 (H)	29.0	1	++	+	−	+	++	++	+++
	16	8	10.0	1	+	−	−	−	−	−	−
	22	29	17.2	3	+	++	+	++	−	+	−
	17	44	3.8	0	+	−	−	−	+++	+	±
	34	49	4.0	0	±	−	−	−	−	−	±
	33	51	9.0	2	±	−	−	−	−	±	±
Cardiac Arrest 5% KCL	31	13	19.0	2	++	−	−	++	−	±	−
	30	14	4.0	0	±	−	−	−	−	±	−
	28	38	26.0	3	+	±	±	−	−	±	−
Cardiac Arrest 20% KCL	23	10 (H)	19.5	4	++	−	−	+	−	++	++
	26	12 (H)	19.0	0	+	−	−	−	+++	+	−
	25	48 (H)	26.0	4	+	−	−	−	−	+	±
	24	22	9.0	4	±	−	−	−	±	++	±
Inflow and Outflow Occlusion	54	0	6.0	2	+	++	−	−	+	+	±
	52	6	6.0	2	++	+	++	−	−	+	++
	51	7	0.0	0	±	−	−	−	−	−	−
	55	7	16.0	2	++	++	++	+	−	+	±
	53	20	4.5	1	++	−	−	−	−	+	±

(B) Cases of cardiac occlusion for 15 minutes under hypothermia (Table 5)

(i) Group of ventricular fibrillation

Either in cases of late death or of long term survival, in which cardiac resuscitation was achieved with difficulty (No. 18 and 22), pathological changes of cardiac tissue were striking: in the former, slight epicardial thickening with hemorrhage and granulocyte infiltration, myocardial degeneration (rarefaction of nuclei and cloudy swelling of protoplasm), muscle necrosis with granulocyte infiltration, fragmentation of muscle fibers and hemorrage extending between them (Fig. 13); in the latter, in addition to epicardial thickening and myocardial degeneration (pycnosis and poorly stained cells), extensive muscle necrosis with calcification and inflammatory reaction (Fig. 14). In the other cases which were defibrillated more easily, histological changes of cardiac tissue were far slighter.

(ii) Group of cardiac arrest

Despite of relative difficulty of cardiac resuscitation, myocardial necrosis was undemonstrable in most cases. In this group, too, the extent of myocardial damage

was proportional to the difficulty of cardiac resuscitation. In No. 23 which was resuscitated with difficulty, intensive fragmentation of muscle fibers and hemorrhage extending between them were found (Fig. 15.).

(iii) Group of inflow and outflow occlusion

In one case (No. 51) which recovered without cardiac resuscitation after release from occlusion, slight epicardial thickening with granulocytes infiltration was found, but no myocardial damage (Fig. 16.). However, in No. 54, 52 and 55 which were defibrillated, myocardial degeneration and necrosis were proved (Fig. 17).

(iv) Résumé

In cases of cardiac occlusion for 15 minutes under hypothermia, divided into the three kinds of occluding method, the more difficult was achieved cardiac resuscitation, the more strikingly were proved histological changes of cardiac tissue, particularly of the myocardium. Above all, in some cases undergoing cardiac massage of longer time, marked fragmentation of muscle fibers and hemorrhage extending among them were found. In the cases of inflow and outflow occlusion, without necessity of cardiac resuscitation, abnormal findings of myocardium were almost undemonstrable, but in the most cases with it due to ventricular fibrillation occuring spontaneously, muscle necrosis was found, in spite of the fact that cardiac resuscitation was achieved more easily in this group than the other two groups. Except the cases of inflow and outflow occlusion which were restored without cardiac resuscitation, the extent of myocardial damage, especially necrosis, was found in the following order; inflow and outflow occlusion, ventricular fibrillation and cardiac arrest. It seems that this fact is due to the grade of interrupting effect of each occluding method upon the blood supply to the heart, which may cause locus minoris resistentiae in myocardium. However, in order to acquire a bloodless operative field, inflow and outflow occlusion had to be carried out at the time of right atriotomy or ventriculotomy, and outflow occlusion at the time of left atriotomy, even when ventricular fibrillation or cardiac arrest was induced. These matters will be discussed in next place.

(C) Cases of various kinds of cardiotomy under hypothermia (Table 6)

As above mentioned, occlusion time was 4 to 9 minutes on this occasion.

(i) Group of ventricular fibrillation

In this group, in general, among cases which were resuscitated by almost the same grade of cardiac resuscitation, the cases of late death had more severe myocardial damage compared with the cases of long term survival. For instance, in No. 42 in which cardiac massage of the longest time in this group was used, all kinds of change from degeneration to necrosis extending over myocardium were demonstrable, in addition to granulocytic cell infiltration, intermuscular hemorrhage and fragmentation of muscle fibers. Some parts of microscopic findings of cardiac tissue in No. 79 and 37 are shown in Fig. 18 and 19.

(ii) Group of cardiac arrest

In No. 45, 87 and 86 which were resuscitated more difficult compared with the other cases in this group, pathological findings of myocardium, especially degenera-

Table 6. Histological Findings of Myocardium Resulting from the Cardiotomies under the 3 Kinds of Occluding Method for 4 to 9 Minutes in Hypothermic Dogs

Cardiac Occlusion	Dog No.	Car App	Survival Time (Day)	Occulusion Time (min.)	Massage Time (min.)	Counter Shock (Frequency)	Myocardium						
							Deg	Nec	Cal	Gra Inf	Hem	Abn Arr	Fra
	79	RV	12 (H)	6.4	2.0	1	++	+	±	−	±	++	+
	82	RV	30 (H)	8.7	6.0	2	++	−	+	+	−	+++	−
Ventricu-	42	RA	48 (H)	5.0	12.0	1	+++	+++	−	++	++	+++	+++
lar	43	RA	4	5.0	3.5	1	+	−	−	+	−	−	−
Fibrill-	49	RV	5	7.0	3.0	1	+	−	−	−	−	−	−
ation	48	RA	8	5.0	2.5	1	++	++	−	++	−	+	−
	47	RA	11	4.7	2.5	1	+	−	−	−	−	−	−
	37	LA	43	4.0	6.0	1	+	−	−	±	−	+	+
	84	RV	12 (H)	9.7	2.0	1	+	+	−	±	+	++	+
Cardiac	45	RA	24 (H)	7.0	25.0	5	+	+++	−	−	++	+	−
Arrest	88	RA	9	8.5	4.0	1	±	−	−	−	−	−	−
5% KCL	50	RV	10	4.7	5.5	2	+	−	−	+	−	+	−
	87	RA	10	7.0	24.0	11	+++	++	+	+	−	++	+
	86	RV	10	8.4	19.0	9	+++	++	+	+	−	++	+
	77	LA	0	5.3	4.3	3	+++	++	−	−	±	−	−
	57	RV	12 (H)	6.3	0.0	0	+	−	−	−	+	++	−
	89	RV	3	9.0	6.2	1	+++	++	−	−	−	+	+
Inflow	60	LA	5	4.0	2.4	1	++	+	+	+	−	++	+
and	56	RV	6	7.3	2.0	1	+++	+++	+	+++	++	+++	+++
Outflow	62	RA	9	4.5	0.0	0	±	±	−	+	−	+	+
Occlusion	61	RA	12	5.3	0.0	0	±	−	−	±	−	+	−
	59	LA	43	4.0	2.5	1	++	+	+	±	−	++	+
	76	RA	97	5.0	1.8	1	++	−	−	−	−	+	−
	75	RA	98	6.5	3.0	1	++	−	−	−	−	+	−

Key : Car App……Cardiac Approach RA……Right Atriotomy
RV……Right Ventriculotomy LA……Left Atriotomy

tion and necrosis, were more striking in degree (Fig. 20).

(iii) Group of inflow and outflow occlusion

In the cases which had no ventricular fibrillation and then did not need cardiac resuscitation, only minimal myocardial degeneration was found. However, in all the cases which underwent cardiac massage and countershock due to occurrence of ventricular fibrillation, moderate or intense myocardial degeneration and in most of them, marked muscle necrosis were proved (Fig. 21).

(iv) Résumé

In all the cases which underwent cardiac approach under inflow and outflow occlusion and recovered without cardiac resuscitation, pathological changes of cardiac tissue were the slightest. Except these cases, there was no significant dfference in the extent of myocardial damage between each kind of occluding method.

IV. DISCUSSION

Either under normothermia or under hypothermia, the cardiac resuscitation

was far more easily accomplished and the mortality rate was lower in the cases of inflow and outflow occlusion than in those of ventricular fibrillation or cardiac arrest.

From the experimental data, the maximum safe time of cardiac occlusion under normothermia may be considered to be about 4 minutes, but this period of the 2 occluding methods except inflow and outflow occlusion resulted sometimes in definite cerebral damage. This fact revealed that the difficulty of cardiac resuscitation had an important connection with the occurrence of cerebral damage. In this view the inflow and outflow occlusion may be considered the most beneficial method of cardiac occlusion.

In 3 of 5 cases subjected to cardiac approach with the similar occlusion for 4 to 9 minutes under hypothermia, developed ventricular fibrillation during these periods or immediately thereafter, but all these cases were resuscitated successfully and their prognosis was good. This fact is similar to the reports of Kay and Gaertner[7] or Shumway and Lewis,[17] confirming that ventricular fibrillation which developed spontaneously at the time of cardiac approach under hypothermia was successfully converted to a normal rhythm.

Even when ventricular fibrillation and cardiac arrest were artificially induced, it was difficult at open heart procedure to prevent profuse bleeding and to acquire a bloodless operative field without inflow and outflow occlusion or only outflow occlusion. Therefore, these managments do not simplify the operative manipulation.

It was proved, in general, that the extent of histological changes of cardiac tissue was proportional to the difficulty of cardiac resuscitation. Only several minutes of inflow and outflow occlusion under normothermia resulted in focal myocardial necrosis, to say nothing of degeneration. This fact shown that the myocardium is a tissue hardly resistant to the interruption of blood supply. Under hypothermia, even more longer period of occlusion resulted in merely the slightest myocardial degeneration, but when ventricular fibrillation developed spontaneously, more severe myocardial damage was produced by cardiac massage and countershock. According to the experiments of Tedeschi and White,[20] even only ventricular fibrillation produced vascular congestion, hemorrhage, edema and muscle fragmentation, and though massage of the normally beating heart produces minimal nonspecific damage, when the fibrillating heart is subjected to manual compression, the changes are somewhat more severe than those observed after fibrillation alone and occasional focal myocardial necrosis results, and moreover countershock produced both epicardial and myocardial damage at the site of application of the electrodes. Peddie, Greech and Halpert[13] expressed the influence of massage in clinical cases, as follows: this manipulation, independent of its duration or technique, produced a progressive pericarditis and moderate to severe myocardial damage, when it was prolonged over fifteen minutes. Consequently it is an important matter to prevent ventricular fibrillation which develops often at intracardiac surgery in the hypothermic state.

It is easy to understand that cardiac arrest is ideal for the purpose of cardiac

occlusion, if the heart is converted to normal rhythm by using any means which is harmless for cardiac tissue. Moulder, Thompson, Smith and Adams[11] demonstrated experimentally that cardiac arrest induced by acetylcholine was easily reversible with atropine, and applied this method for 5 clinical cases. Young, Sealy, Brown, Hewitt, Callaway, Merritt and Harris[20] induced cardiac arrest by using cardioplegic agent and restored cardiac activity by coronary perfusion with oxygenated blood. Further development of these methods will be expected.

The pupils of all dogs which were subjected to cardiac occlusion, dilated extremely after a certain time and contracted gradually to the original size after release from occlusion with or without cardiac resuscitation. Even though the heart was converted to a normal rhythm, the cases of which the pupils did not contract or dilated again after transitory contraction, were absolutely bad in prognosis. Thus the recovery of cardiac function was proportional to the grade of pupil contraction. Bellville, Artusio and Glenn[1] recommended to apply the electroencephalogram as a monitor to determine the effective output during cardiac surgery, as the brain is the most sensitive organ to oxygen lack. In our experiments, inflow and outflow occlusion resulted in deterioration of electroencephalographic activity and then dilatation of the pupils, while release from occlusion resulted in contraction of the pupils and then return of electroencephalographic activity. However, it seemed, under hypothermia, to take a fairly long time for electrical activity to return. This fact means that the cerebral cortex is more sensitive to oxygen lack than the oculomotor nuclei which regulate the pupilar sphincters. According to Hirai, Endo and Saito,[4] the times required for electrical activity to return after release from temporary circulatory occlusion to the brains of hypothermic dogs, are concerned with periods of occlusion, body temperatures of the animals and the times elapsed until rewarming, which promotes markedly recovery of electrical activity. In our experiments, inflow and outflow occlusion for about 5 minutes under hypothermia was followed by incomplete recovery of EEG pattern even after 15 minutes due to no rewarming. The above mentioned matters reveal that the pupil size is useful for decision of cardiac function.

V. SUMMARY

I. We have carried out experimental studies to learn whether ventricular fibrillation and cardiac arrest induced, respectively, electrically and chemically can be used for intracardiac surgery under direct vision.

2. We cannot support the clinical application of the artificially induced ventricular fibrillation and cardiac arrest for the following reasons.

(i) The maximum safe time of cardiac occlusion, i. e., a) ventricular fibrillation, b) cardiac arrest and c) inflow and outflow occlusion, under normothermia was about 4 minutes, but even for this time definite cerebral damage developed in some cases of a and b), and the best result was obtained by the method c).

(ii) The experiments of cardiac occlusion for 15 minutes and cardiac approach with the same occlusion for 4 to 9 minutes under hypothermia revealed that the

easiest recovery of the heart and the lowest mortality were obtained by the method c).

(iii) In performing a cardiotomy, inflow and outflow occlusion (a right atriotomy and ventriculotomy) or outflow occlusion (a left atriotomy) had to be carried out in order to prevent profuse bleeding and to acquire a bloodless operative field, even when the methods a) and b) had been used.

(iv) Air embolism by which a cardiotomy with the method c) is apt to be followed, was able to be avoided by pouring a saline solution sufficiently into the cardiac cavity through the incision at suturing.

(v) Even ventricular fibrillation which developed spontaneously in the hypothermic state, was easily converted to a normal rhythm and its prognosis was not always bad.

(vi) Cardiac resuscitation (massage and countershock) which is always necessary in the cases of a) and b), damaged cardiac tissue sometimes seriously.

3. Only inflow and outflow occlusion for 4 to 6 minutes under normothermia resulted in moderate myocardial degeneration and necrosis.

4. Inflow and outflow occlusion for 15 minutes and cardiac approach with the same occlusion for 4 to 9 minutes under hypothermia resulted in only slight myocardial degeneration, when they were not associated with ventricular fibrillation, that is to say, when cardiac resuscitation which produced moderate to severe myocardial degeneration and focal muscle necrosis even if it was achieved easily, was not used.

5. It was revealed that the recovery of cardiac function was proportional to the grade of pupilar contraction, and there was a certain correlation between changes of pupil size and those of electroencephalographic pattern following cardiac occlusion and release from it. Accordingly, the pupil size is useful for decision of cardiac function.

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Explanation of Photomicrographs

Fig. 10. Cloudiness of protoplasma andsmall focalmuscle necrosis (an arrow), from dog No. 65 which was subjected to cardiac arrest for 5 minutes under normothermia and dide immediately after operation. Cardiac resuscitation was achieved with difficulty (13 minutes of massage and 6 times of countershock in amount).

Fig. 11. Perinuclear vacuolization, from the same dog as in Fig. 10.

Fig. 12. Focal myocardial necrosis with calcification, from an animal (No. 69) subjected to 4 minutes of inflow and outflow occlusion under normothermia and sacrificed 8 days after the procedure. No cardiac resuscitation was needed

Fig. 13. Fragmentation of muscle fibers and hemorrhage extending between them with granulocyte infiltration, from a heart (No. 18) subjected to ventricular fibrillation for 15 minutes under hypothermia. The animal was resuscitated by manual compression for 29 minutes and one time of countershock and survived for 5 hours.

Fig. 14. Extensive myocardial necrosis with inflammatory reaction. The animal (No. 22) was subjected to the same manipulation as carried out in dog No. 18 and sacrificed 29 days later. Defibrillation was achieved by using massage for 17.2 minutes and 3 times of countershock.

Fig. 15. Myocardial degeneration (Pycnosis, vacuolization of nuclei and homogenous staining of protoplasm), from a dog (No. 23)subjected to 15 minutes of cardiac arrest in the hypothermic state. The animal was resuscitated by using cardiac massage for 19.5 minutes and 3 times of countershock and survived for 10 hours.

Fig. 16. Myocardium showing almost normal appearance, from a dog (No. 51) subjected to inflow and outflow occlusion for 15 minutes under hypothermia and sacrificed 7 days later. No cardiac resuscitation was necessary.

Fig. 17. Focal myocardial necrosis with calcification, from dog No. 52 subjected to the same manipulation as carried out in No. 51 and sacrificed 6 days after operation. Cardiac resuscitation (manual compresssion for 6 minutes and 2 times of countershock) was done due to spontaneous development of ventricular fibrillation.

Fig. 18. Interstitial myocardial edema and intermuscular hemorrhage, from a dog(No. 79) subjected to a right ventriculotomy under induced ventricular fibrillation for 6.4 minutes in the hypothermic state. Defibrillation was achieved easily (massage for 2 minutes and 1

time of countershock). The animal survived for 12 hours.

Fig. 19. Epicardial thickening and subepicardial hyalin degeneration of myocardium, from an animal (No. 37) subjected to a left atriotomy under induced ventricular fibrillation for 4 minutes in hypothermic state and sacrificed 43 days later. Defibrillation was chieved by using massage for 6 minutes and 1 time of countershock.

Fig. 20. Subepicardial necrosis of myocardium, from a dog (No. 45) subjected to a right atriotomy under cardiac arrest for 7 minutes in the hypothermic state and resuscitated by using 25 minutes of manual compression and 5 times of countershock in amount. twenty-four hours of survival were allowed.

Fig. 21. Extensive myocardial necrosis with calcification and inflammatory reaction, from dog No. 56 subjected to a right ventriculotomy under inflow and outflow occlusion in the state of hypothermia and sacrificed 6 days after operation. The fibrillating heart was converted to a normal rhythm by using massage for 2 minutes and 1 time of countershock.

和文抄録

直視下心内手術に於ける人為的心室細動及び心搏停止の応用に関する実験的研究

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1. 電気刺激により誘発した心室細動及びKcl溶液の左心室内注入によつて生ぜしめた心搏停止が直視下心内手術に於て実際に用いる価値があるか否かを実験的に検討した。

2. 次の理由により上記の人為的心室細動及び心搏停止を直視下心内手術で応用することには賛成し難い。

(i) 常温下長期生存例を得る心血流遮断（a. 心室細動，b. 心搏停止，c. Inflow及びOutflow閉鎖）の時間的限界は大体4分で，この程度の遮断でもa及びbによつたものには脳障害を来たしたものがあり，cの方法が最も心臓の回復が容易且つ良好である。

(ii) 全身冷却下15分の心血流遮断実験でも，4～9分の遮断で更に各種の心切開（右房，右室，左房）を加へた場合でもcの方法を行つた時が最も心蘇生が容易で生存率も高い。

(iii) 心臓切開を行う場合aまたはbの方法を行つても，多量の出血を防止し無血手術野を得る為には，右房又は右室切開時にはInflow及びOutflow閉鎖を，左房切開時にはOutflow閉鎖を行わねばならない。

(iv) cの方法はa，bの方法に比較して心切開，特に左房切開の際冠動脈空気栓塞を起し易いが，切開創閉鎖に際し生理的食塩水を充分心内に注入することにより予防し得る。

(v) 全身冷却下で心血流を遮断したため，或は更に心切開を加えたために誘発される心室細動は容易に除去されその予後は悪くない。

(vi) a，bの方法を行つた場合，必ず心蘇生術（心臓マッサージ及びCounter-Shock）を必要とするが，これは甚だしく心組織を障害することがある。

3. cの方法で心血流を遮断した場合常温下では4～6分の遮断のみで相当の心筋変性と壊死を来すが，一方全身冷却下の15分間の遮断実験及び心切開実験では心室細動を誘発しなかつたもの，即ち心蘇生術を要しなかつた例では軽度の心筋変性を認めたに過ぎなかつたが，これを必要としたものでは中等度から強度の心筋変性のみならず壊死をも認めた．従つて全身冷却下に直視下心内手術を行う際は心室細動を未然に防止する様努力すべきである。

4. 犬の瞳孔は常温下でも全身冷却下でも心血流遮断により徐々に散大し，或る時間後には極度に散大するが，心蘇生術により縮少して元に還る．心蘇生術により心臓が正常の調律を回復しても，瞳孔の縮少しないもの，又は一旦縮小しても再び散大するものは予後絶対に不良であつた．又瞳孔は心血流遮断によつて脳波の変化に遅れて散大し始め，脳波が消失してから極度に散大し，蘇生術により先ず瞳孔が縮少して元に還へり次いで脳波が回復した．従つて瞳孔の大きさは心機能の判定即ち有効心搏出量の決定に役立つ。

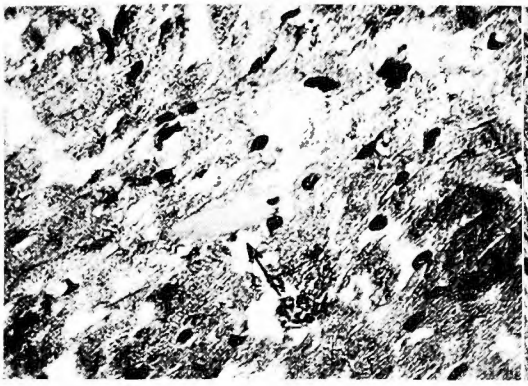

Fig. 10

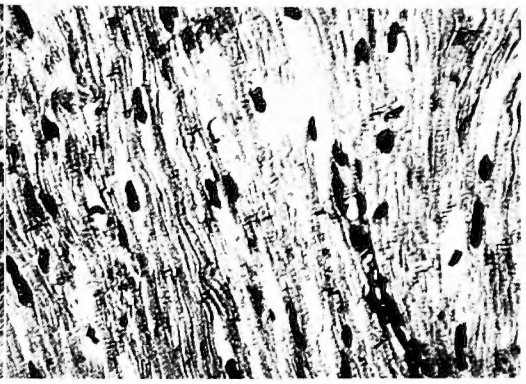

Fig. 11

Fig. 12

Fig. 13

Fig. 14

Fig. 15

Fig. 16

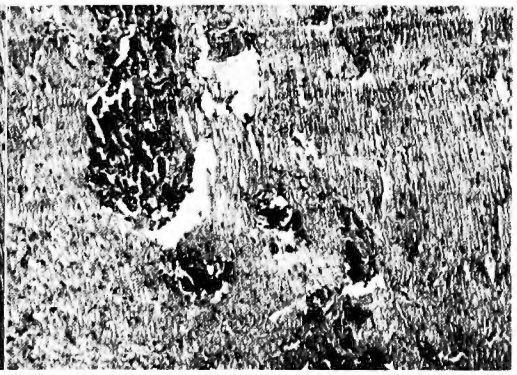

Fig. 17

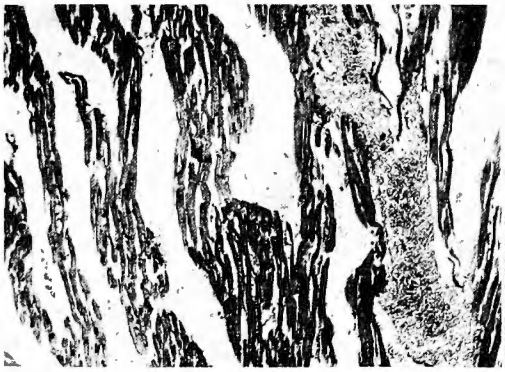

Fig. 18

Fig. 19

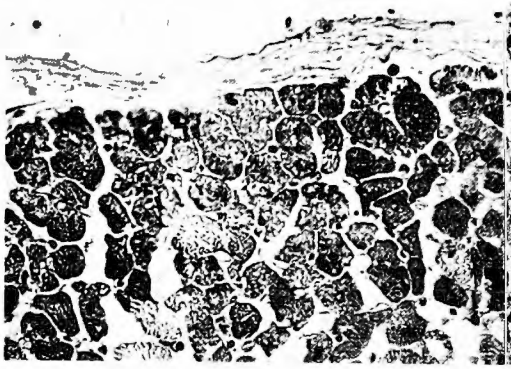

Fig. 20

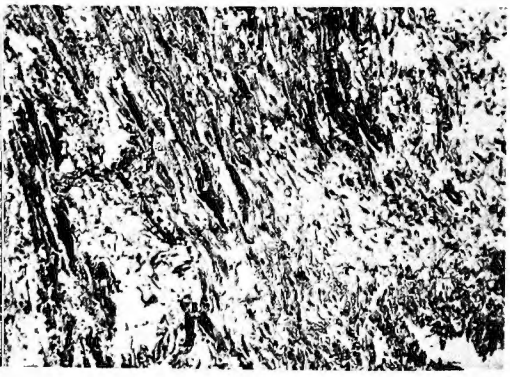

Fig. 21